Cardiovascular Risk Factors in People Older Than 65

RISK FACTORS for the development of coronary heart disease and atherosclerosis have been well established for the middle-aged population. Only recently, however, has convincing evidence shown that all of the following are independent cardiovascular predictors in persons 65 years and older: total serum cholesterol, high- and low-density-lipoprotein cholesterol (HDL and LDL) and triglyceride levels, hypertension, left ventricular hypertrophy, glucose levels, physical inactivity, obesity, and cigarette smoking. These conclusions are derived from multivariate analyses of large, well-designed prospective studies, including the 30-year data from the Framingham Heart Study and the 12-year data from the Honolulu Heart Program.

The ratio of total and HDL cholesterol values and the HDL levels are much better predictors of coronary heart disease in elderly men and women than are LDL, total cholesterol, or triglyceride levels. The desirable ratio for men is less than 4.0 and for women is less than 3.5. Triglyceride is also a strong predictor in women and in some subsets of men. The combination of high triglyceride levels (greater than 150 to 200 mg per dl) and low HDL (less than 35 to 40 mg per dl) levels may represent a high-risk lipid profile even if the total cholesterol value is only 200 mg per dl. These findings suggest that, in patients older than 65, HDL is the most important lipid for predicting cardiovascular risk (more so than LDL) and that triglyceride levels are useful for further coronary heart disease risk stratification.

This information is an important "amendment" to both the 1987 National Cholesterol Education Program Expert Panel's and the 1983 National Institutes of Health Triglyceride Consensus Panel's lipid-lowering guidelines, which primarily emphasized coronary heart disease risk with LDL levels higher than 130 mg per dl and triglyceride levels higher than 250 mg per dl, respectively. Although no prospective studies to date have tested whether raising HDL levels is protective or whether HDL functions independently from LDL or triglyceride, studies have recently shown possible atherogenic mechanisms associated with hypertriglyceridemia, such as a positive correlation with small, dense, atherogenic LDL particles and a negative correlation with HDL levels. There is also an association between hyperinsulinemia and hypertriglyceridemia that may play a role in atherogenesis. Lipid-lowering dietary studies have shown significant reductions in CHD morbidity and mortality in the elderly, and clinical trials are under way to determine the efficacy of hypolipidemic drug therapies. Until such data become available, it is generally considered prudent, in addition to following the amended National Cholesterol Education Program guidelines, to recommend hygienic measures that raise HDL levels (and lower triglyceride levels), such as smoking cessation, weight reduction, and increased physical activity. Furthermore, the choice of all medications used in these patients should be evaluated for their favorable effect on HDL levels; secondary causes of hypertriglyceridemia (such as alcoholism, diabetes mellitus, and drugs) should always be excluded.

Isolated systolic hypertension (blood pressure 160 mm of mercury or higher), diastolic hypertension (blood pressure 90 mm of mercury or higher), and left ventricular hypertrophy are all independent risk factors for coronary heart disease in those older than 65. Left ventricular dysfunction, leading to congestive heart failure, is a leading cause of disability in this age group, which has a high prevalence of hypertension. Many well-designed controlled trials, such as the European Working Party on High Blood Pressure in the Elderly, have shown substantial reductions in cardiovascular mortality resulting from the treatment of combined systolic and diastolic hypertension. Most recently, the Systolic Hypertension in the Elderly Program study, which involved medically treating isolated systolic hypertension, has shown a significant reduction in total stroke incidence. Reductions in other major events were also noted.

Cigarette smoking—which is known to decrease HDL levels—increases the coronary heart disease risk in the elderly, especially if more than 40 cigarettes are smoked per day. Elevated blood glucose levels (greater than 130 mg per dl) are a major risk factor for the disease in men and women. Although the correlation is not as strong, both obesity (as defined by a relative weight of 130% or more by Metropolitan Life Insurance tables) and physical inactivity increase the risk of coronary heart disease.

Persons older than 65 constitute more than 12% of the United States' total population. They use 33% of all health care costs, most of which is directed toward the treatment of cardiovascular diseases. Encouraging international data have shown that significant reductions in coronary heart disease morbidity and mortality rates are occurring in older patients, even in those older than 74 years.

The Stockholm Ischaemic Heart Disease Secondary Prevention Study has even shown a reduction in overall mortality using lipid-lowering dietary and drug therapy. Advances in the understanding of cardiovascular risk factors and their contribution to an increased prevalence of coronary heart disease should encourage greater efforts toward modifying risk factors and possibly improving the quality of life in people older than 65.

KEITH R. DORAM, MD
GREGORY R. WISE, MD
Loma Linda, California

REFERENCES

Aronow WS: Cardiac risk factors: Still important in the elderly. Geriatrics 1990; 45:71-74, 79-80

Benfante R, Reed D: Is elevated serum cholesterol level a risk factor for coronary heart disease in the elderly? JAMA 1990; 263:393-396

Carlson LA, Rosenhamer G: Reduction of mortality in the Stockholm Ischaemic Heart Disease Secondary Prevention Study by combined treatment with clofibrate and nicotinic acid. Acta Med Scand 1988; 223:405-418

Castelli WP, Wilson PW, Levy D, Anderson K: Cardiovascular risk factors in the elderly. Am J Cardiol 1989; 63:12H-19H

Gordon DJ, Rifkind BM: Treating high blood cholesterol in the older patient. Am J Cardiol 1989; 63:48H-52H

SHEP Cooperative Research Group: Prevention of stroke by antihypertensive drug treatment in older persons with isolated systolic hypertension—Final results of the Systolic Hypertension in the Elderly Program. JAMA 1991; 265:3255-3264

Benefits of Dual-Chamber Pacemakers

PACING THERAPY has progressed rapidly over the past 33 years from a major surgical procedure requiring a thoracotomy to one that can be safely and effectively performed at a community hospital under local anesthesia. There has been a concomitant increase in the understanding of cardiac physiology and the capabilities of implanted pacemakers. Pacemakers today are capable of pacing and sensing in both chambers of the heart to restore the normal coordination between the atria and ventricles while modulating the rate in

accord with physiologic requirements. These are called dual-chamber rate-modulated systems and are identified by the code DDDR. The increased complexity of these systems has led to difficulty interpreting electrocardiographic rhythms, an increased requirement for follow-up, and increased cost. Given these factors, it must be asked if these technologic advances are worth the additional costs. Because it is expected that a pacemaker will function properly for five to ten years, the long-term consequences of a pacing mode need to be considered when evaluating the cost.

Recent studies have shown that the maintenance of atrioventricular synchrony is associated with a dramatic reduction in the incidence of atrial fibrillation, systemic emboli, the development of overt congestive heart failure, and even mortality. In addition, adverse hemodynamic consequences of single-chamber (code VVI) pacing, originally thought to have an incidence of only 10% to 15%, are now being found in more than 80% of VVI-paced patients. The lower number reflects severe manifestations of the pacemaker syndrome that include overt congestive heart failure and syncope. The higher number identifies symptoms for which the body can compensate or with which the patient can learn to live but nevertheless affect the quality of life. These include palpitations caused by canon A waves resulting from the atrium contracting against a closed atrioventricular valve, increased mitral and tricuspid regurgitation, a nonproductive cough, symptoms of low cardiac output, and decreased cerebral perfusion resulting in an unsteady gait and periods of increased confusion. These symptoms may be intermittent.

The goal of treating a cardiac rhythm abnormality is to restore normal sinus rhythm. Sinus rhythm supports the heart rate at rest and accelerates with exercise while at all times maintaining synchrony between the atrium and ventricles. Dual-chamber rate-modulated pacing is the closest that technologic advances have come to mimicking normal sinus rhythm. Despite the increased costs associated with implanting the device, studies are documenting both the short- and long-term benefits of dual-chamber pacing. If the costs associated with repeated hospital admissions are considered—increased medication requirements and the quality of life associated with the late consequences of single-chamber pacing—dual-chamber pacing improves a patient's quality of life and becomes more cost effective. Several studies have shown that there is no longer justification to implant a single-chamber pacemaker in every patient who requires pacing therapy. The American College of Cardiology and American Heart Association guidelines state that

> long-term absence of atrioventricular synchrony increases the incidence of atrial fibrillation and stroke and may reduce patient life expectancy. . . . therefore, the concept that the single chamber pacemaker with adaptive-rate functions is equivalent to the dual chamber pacemaker cannot be supported.

PAUL A. LEVINE, MD
Loma Linda, California

REFERENCES

Clarke M, Sutton R, Ward D, et al: Pacemaker prescription for symptomatic bradycardia—Report of a working party of the British pacing and electrophysiology group. Br Heart J 1991; 66:185-191

Dreifus LS, Fisch C, Griffin JC, Gillette PC, Mason JW, Parsonnet V: ACC/AHA Task Force report, guidelines for implantation of cardiac pacemakers and antiarrhythmia devices. J Am Coll Cardiol 1991; 18:1-13

Heldman D, Mulvihill D, Nguyen H, et al: True incidence of pacemaker syndrome. PACE 1990; 13:1742-1750

Tyers GF: Current status of sensor-modulated rate-adaptive cardiac pacing. J Am Coll Cardiol 1990; 15:412-418

Zanini R, Facchineti AI, Gallo G, Cazzamalli L, Bonandi L, Deicas L: Morbidity and mortality of patients with sinus node disease: Comparative effects of atrial and ventricular pacing. PACE 1990; 13:2076-2079

Diabetic Nephropathy Revisited

DIABETIC NEPHROPATHY is the leading cause of end-stage renal disease in patients with diabetes mellitus and is the single most frequent reason in the United States for starting long-term hemodialysis treatment. Diabetic nephropathy is defined by persistent proteinuria on a dipstick test (or greater than 0.3 grams per day urinary albumin excretion) in the absence of other renal disease. The prevalence of end-stage renal disease caused by diabetes ranges from less than 10% in patients with type II diabetes to about 40% in those with type I diabetes. Nonetheless, some groups with type II diabetes—such as African Americans, Asian Americans, Hispanic Americans, and Native Americans—have a much higher risk of end-stage renal disease than expected. Furthermore, diabetic patients with a family history of diabetic nephropathy or hypertension are at increased risk of nephropathy developing.

Therapeutic and preventive measures both aim at delaying the progression of diabetic nephropathy to end-stage renal disease. The control of hyperglycemia to "near normal" (tight control) is often considered an important first step in attempting to slow the progression of microalbuminuria to renal failure. Some renal mesangial changes regress with tight control and pancreatic transplantation. Although debate continues about the merits of tight control, poor glycemic control does strongly predict clinical diabetic nephropathy in diabetes. Once nephropathy is established, the control of blood glucose is ineffective in reversing the process.

Blood pressure control in patients with diabetes dramatically reduces the albumin excretion rate and slows the progression of renal failure. A loss of renal mass, as can occur in diabetic patients, results in compensatory glomerular hyperfiltration in the remaining nephrons. Although angiotensin-converting enzyme (ACE) inhibitors and some calcium channel blockers reduce the glomerular hyperfiltration, ACE inhibitors may worsen renal function and induce or worsen hyperkalemia in some patients.

Protein-restricted diets show promise in decreasing glomerular hyperfiltration and may play a role in therapy and prevention. Some studies show that such diets can decrease the urinary albumin content and delay the decline of creatinine clearance. Whether this results in the prevention of clinical renal failure over the long term remains controversial.

Other interventions proposed to slow the rate of progression of chronic renal failure in diabetic patients include platelet (thromboxane synthesis) inhibitors; phosphate-restricted, low-protein diets; vegetarian (no animal protein) diets; and aldose reductase inhibitors. The role of these interventions should be clarified in this decade.

In summary, diabetic nephropathy is a serious and costly problem. End-stage renal disease does not develop in all diabetic patients; why it does in some and not others is an enigma. Some preventive strategies are well established, and the aggressive control of even mild hypertension is essential. The increasingly diversified group of proteinuria-sparing drugs makes it easier to individualize care. It is prudent to maintain tight glycemic control, especially in patients with type I diabetes and in high-risk subpopulations of those with type II diabetes. A cautious use of any potentially nephrotoxic medication or intravenously administered contrast media is warranted. Protein restriction is highly advisable. Screening for proteinuria is recommended because it may